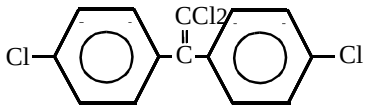
DDE
(p,p-DICHLORODIPHENYLDICHLOROETHYLENE)

DDE is a federal hazardous air pollutant and was identified as a toxic air contaminant in April 1993 under AB 2728.

CAS Registry Number: 72-55-9

Molecular Formula: $C_{14}H_8Cl_4$



DDE (p,p-Dichlorodiphenyldichloroethylene) is a white, crystalline solid. It is soluble in water (at 0.010 parts per million), fat, and most organic solvents. It is a degradation product of dichlorodiphenyltrichloroethane (DDT) and is found as an impurity in DDT (HSDB, 1991).

Physical Properties of DDE

Synonyms: dichlorodiphenyldichloroethylene; 1,1'-dichloroethenylidene-bis(4-chlorobenzene); DDT dehydrochloride; p,p'-DDE; 2,2-bis(4-chlorophenyl)1,1-dichloroethene

Molecular Weight:	318.0
Melting Point:	88.4 °C
Vapor Pressure:	6.5×10^{-6} torr at 20 °C
Log Octanol/Water Partition Coefficient:	6.51
Conversion Factor:	1 ppm = 13.0 mg/m^3

(HSDB, 1991; U.S. EPA, 1994a)

SOURCES AND EMISSIONS

A. Sources

DDE occurs as an impurity in commercial DDT formulations and as a biodegradation product of DDT. DDT use has been banned in the United States since the early 1970s (HSDB, 1991).

B. Emissions

No emissions of DDE from stationary sources in California were reported, based on data obtained from the Air Toxics “Hot Spots” Program (AB 2588) (ARB, 1997b).

C. Natural Occurrence

DDE is a synthetic organic chemical and does not have any natural sources (HSDB, 1991).

AMBIENT CONCENTRATIONS

No Air Resources Board data exist for ambient measurements of DDE. However, the United States Environmental Protection Agency (U.S. EPA) has compiled ambient air data from several locations throughout the United States. The data reported a mean concentration of DDE of 0.26 nanograms per cubic meter (ng/m^3) in Jacksonville, Florida, with a range from 0.04 to 0.66 ng/m^3 from 1987-88. In 1978, a mean concentration of DDE was measured at 4.5 ng/m^3 with a range of 0 to 14.2 ng/m^3 in Columbia, South Carolina (U.S. EPA, 1993a).

INDOOR SOURCES AND CONCENTRATIONS

No information about the indoor sources and concentrations of DDE was found in the readily-available literature.

ATMOSPHERIC PERSISTENCE

DDE partitions between the gas and particle phases, with Bidleman, et al., 1986, estimating 2 percent and 24 percent of DDE to be particle-associated at 20 ^oC and 0 ^oC, respectively, based on field measurements. The atmospheric half-life of DDE, due to its reaction with hydroxyl radicals, is estimated to be 2 days. DDE is, therefore, expected to be removed from the atmosphere by wet and dry deposition of particle phase DDE and gas phase reaction with the hydroxyl radical (Atkinson, 1995).

AB 2588 RISK ASSESSMENT INFORMATION

DDE emissions are not reported from stationary sources in California under the AB 2588 program. It is also not listed in the California Air Pollution Control Officers Association Air Toxics “Hot Spots” Program Revised 1992 Risk Assessment Guidelines as having health values (cancer or non-cancer) for use in risk assessments (CAPCOA, 1993).

HEALTH EFFECTS

Probable routes of human exposure to DDE are inhalation and ingestion (U.S. EPA, 1994a).

Non-Cancer: The U.S. EPA reports that no information on acute effects in humans through inhalation exposure is available. However, oral exposure to high doses of the parent compound DDT causes central nervous system effects in humans manifested by headaches, nausea, and convulsions. DDE is a metabolite of DDT. The only effect noted in epidemiologic studies of

workers exposed to DDT was an increase in liver enzyme activity, with no adverse effects on the blood, liver, heart, or central nervous system noted. Animal studies have shown some evidence for effects on the liver, immune system, and central nervous system from chronic oral exposure to DDT. The U.S. EPA has not developed a Reference Concentration (RfC) or an oral Reference Dose (RfD) for DDE (U.S. EPA, 1994a).

DDT and DDE have been found in human blood, placental tissue and umbilical cord blood. Levels of DDT, DDE and/or dichlorodiphenyldichloroethane (DDD) were higher in maternal blood or placental tissue of mothers who gave birth prematurely or spontaneously aborted. However, in most studies the difference was not statistically significant, and increased levels of polychlorinated biphenyls (PCBs) and/or other chlorinate pesticides were also found (ATSDR, 1994). Thus, human studies are inconclusive. DDT impaired reproduction and/or development in mice, rats, rabbits, dogs and avian species (IARC, 1991). Specific effects on development have included increased post-implantation loss, reduced fetal weight, increased postnatal mortality, reduced postnatal weight gain, and neurobehavioral effects. Some of these effects may be mediated by lactational exposure to DDT or DDE secreted in milk (ATSDR, 1994b; IARC, 1991c).

Cancer: The U.S. EPA has found studies of workers exposed to DDT have yielded conflicting results. An increased incidence of liver tumors has been reported in mice and hamsters, and thyroid tumors in female rats from oral exposure to DDE (U.S. EPA, 1994a).

The U.S. EPA has classified DDE as Group B2: Probable human carcinogen, and calculated an oral unit risk estimate of 9.7×10^{-6} (microgram per liter)$^{-1}$. The U.S. EPA estimates that, if an individual were to ingest water containing DDE at 0.1 micrograms per liter over his or her entire lifetime, that person would theoretically have no more than a 1 in 1 million increased chance of developing cancer (U.S. EPA, 1994a). The International Agency for Research on Cancer has classified the parent compound DDT as Group 2B: Possible human carcinogen based on sufficient evidence in animals (IARC, 1987a).

The State of California has determined under Proposition 65 that DDE and DDT are carcinogens (CCR, 1996). (DDD is also listed as a carcinogen under Proposition 65 (CCR, 1996)). The inhalation potency factor that has been used as a basis for regulatory action in California for DDE and DDT are 9.7×10^{-5} (microgram per cubic meter)$^{-1}$ (OEHHA, 1994). In other words, the potential excess cancer risk for a person exposed over a lifetime to 1 microgram per cubic meter of DDE or DDT is estimated to be no greater than 97 in 1 million. The oral potency factor that has been used as a basis for regulatory action in California for DDE and DDT is 3.4×10^{-1} (milligram per kilogram per day)$^{-1}$ (OEHHA, 1994).